V. "On the Effects of Increased Arterial Pressure on the Mammalian Heart." By JOHN A. MCWILLIAM, M.D., Professor of the Institutes of Medicine in the University of Aberdeen. Communicated by Professor M. FOSTER, Sec. R.S. Received May 30, 1888.

The following is a short preliminary statement of some of the main facts elicited in the course of a recent investigation. The experiments were conducted on chloroformed cats. The thorax was laid open, artificial respiration being maintained, and the action of the auricles and ventricles was recorded by means of the graphic method. The contraction of the heart in ordinary circumstances having been observed and registered, the arterial pressure was raised by constricting or clamping the last part of the thoracic aorta—usually for a period of 4—8 seconds. Clamping for longer periods was often accompanied by convulsive movements of the animal.

The results may be briefly summarised as follows:—

They fall into one or other of two categories according as to whether the medullary cardio-inhibitory mechanism is (I) functionally active in controlling the heart's action, or is (II) incapable of affecting the cardiac beat. The latter condition is one that may result from various causes, such as (*a*) section of the vagus nerves or paralysis of their function through the influence of drugs, &c.; (*b*) depression or paralysis of the medullary cardio-inhibitory *centre*, brought about by drugs or by other causes.

I. In the first-mentioned condition, when the cardio-inhibitory mechanism is in a position to control the heart's action, a marked rise of the arterial pressure (such as results from compression of the descending aorta) causes, as Marey has shown, a slowing of the cardiac rhythm.

I find that the rise of blood-pressure also causes marked changes in the contraction force of the cardiac muscle. For a short time (a few seconds, 1, 2, 3, &c.), after clamping of the descending aorta there occurs an augmentation in the strength of the beat—especially of the ventricular beat; meanwhile the rhythm has become slower than before (fig. 1).

Then there occurs a more or less sudden change. The auricular contractions undergo a striking diminution in force. They remain enfeebled until the compression of the aorta has been discontinued and the blood pressure has fallen; then they gradually recover, though the process of recovery may not always begin at once (figs. 1 and 2).

The changes in the vertricular action consequent upon closure of

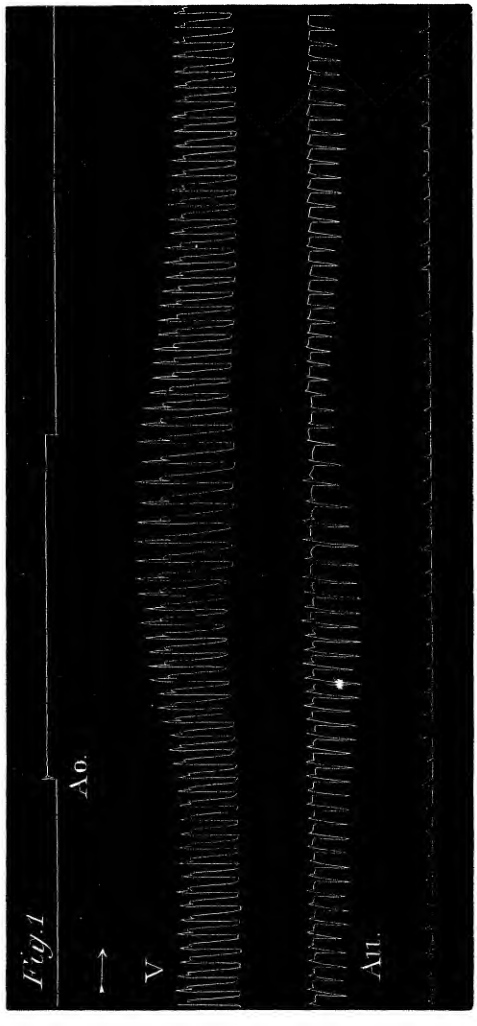


Fig. 1

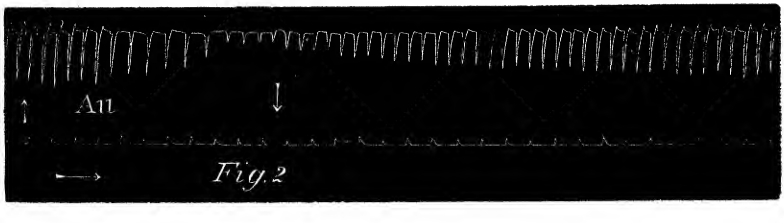


Fig. 2

the descending aorta do not run parallel with those occurring in the auricles. The ventricles, while they beat more slowly than before, usually beat much *more strongly* even when the auricular contraction has become markedly weakened (fig. 1). Depression of the ventricular force may occur, but it comes considerably later than the auricular depression, and is very much slighter in degree (fig. 3). The slow strong ventricular systoles are able to empty the cavity of the left ventricle when systoles of less strength fail to do so—as indicated by the fact that the recording lever often fails to descend to the ordinary level in the interval between the contractions (fig. 1). When the descending aorta has been released and the pressure has fallen, a period of marked cardiac acceleration often succeeds; during this acceleration, the individual ventricular beats are much diminished in force (fig. 3).

The above-mentioned cardiac changes attendant on a sudden rise of arterial pressure are brought about through the medullary cardio-inhibitory centre and the vagus nerves. They are of such a nature that while the ventricles are contracting slowly and powerfully in such a way as to be able to discharge their contents in spite of the increased arterial pressure, there occurs a striking change in the action of the auricles involving a great reduction in the amount of blood pumped into the ventricles and the degree in which the latter are distended just before their systole. Hence the quantity of blood thrown out by the left ventricle into the systemic arteries is much diminished, and the rise in the blood-pressure is in some measure counteracted and controlled.

II. In conditions where the medullary cardio-inhibitory mechanism has ceased to exert any controlling influence upon the heart (*e.g.*, after section of both vagi), the effects following a sudden rise of arterial pressure are entirely different from those above described.

Marey showed that there was no very constant relation between the rate of the heart's action and the height of the blood-pressure after section of the vagi; some degree of acceleration was commonly observed.

Examining the cardiac changes in the way already mentioned, I find that after section of the vagi or paralysis of the medullary cardio-inhibitory mechanism, a sudden rise of arterial pressure causes no very striking or constant change in the heart's rhythm; frequently there is *slight* acceleration. There is a complete absence of the characteristic changes in the contraction force above described (under I). As regards the strength and character of the cardiac action, there are two conditions to be noted.

(1.) The heart may at each systole be able to discharge its contents in normal or approximately normal fashion. In such circumstances the principal change to be observed in a vigorous heart is a marked

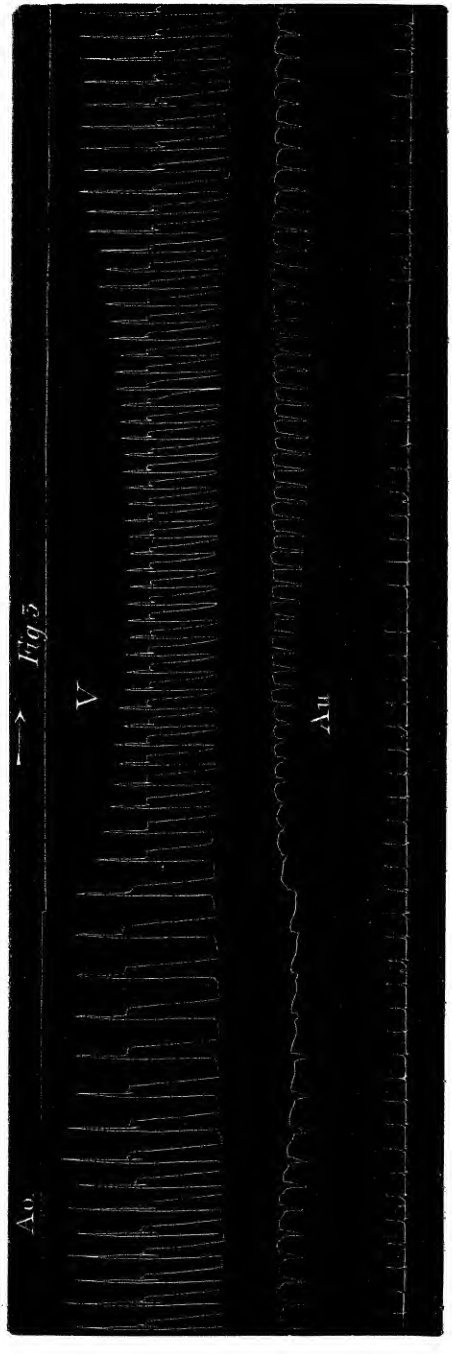
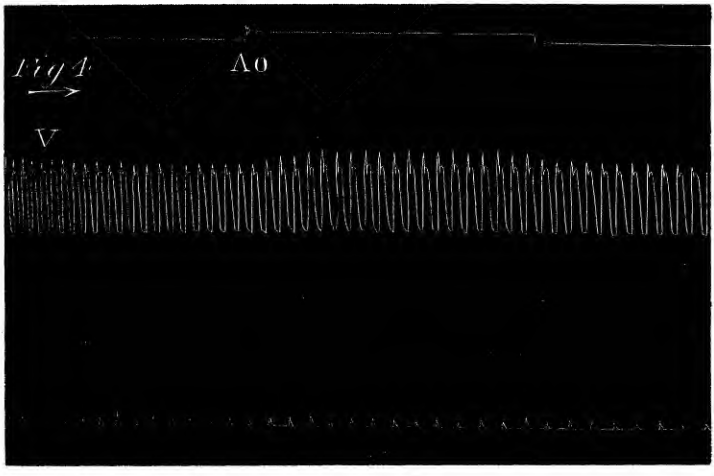


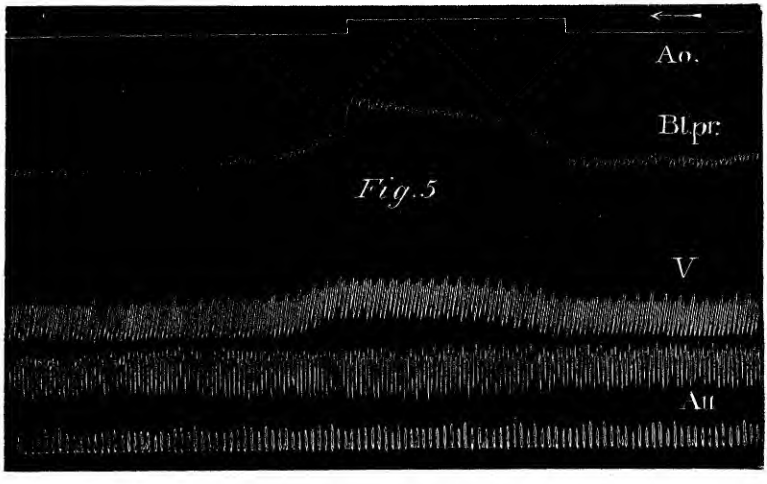
Fig 3

increase in the force of the beat, at least in the ventricles (fig. 4).



(2.) On the other hand the relation of the ventricular power to the arterial resistance may be such that the left ventricle is not able to expel its contents at each beat in the normal fashion. The recording lever fails to descend to the usual level between the contractions; it remains elevated to a considerable extent from the ordinary base line.

The results occurring in both the conditions referred to—(1) and



(2)—are not obviated by section of all the visible branches of the annulus of Vieussens, and of the vago-sympathetic in the neck and thorax. They appear to depend on properties of the heart itself, and not on the influence of extra-cardiac nerves.

DESCRIPTION OF FIGURES.

FIG. 1.—Tracing of auricles and ventricles, showing effects of clamping descending aorta (Ao.). In the ventricular tracing the upward movement indicates contraction; in the auricular tracing the downward movement indicates contraction. The time tracing shows half seconds.

FIG. 2.—Tracing of auricles. Downward movement indicates contraction. Descending aorta clamped at the point marked ↓, and released at ↑. Time marker indicates half seconds.

FIG. 3.—Tracing of auricles and ventricles. In the ventricular tracing contraction is represented by the upward movement, in the auricular tracing by the downward movement. Time marker shows half seconds. Clamping of descending aorta.

FIG. 4.—Tracing of ventricles; upward movement indicates contraction. Increase in size of beats during the closure of the descending aorta. Time marker indicates half seconds.

FIG. 5.—Tracings of auricles, ventricles and blood-pressure in left carotid artery. The lowest tracing marks the time in half seconds. The level of the ventricular tracing rises during closure of the descending aorta; there is incomplete emptying of the left ventricle at each systole.

Presents, June 7, 1888.

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Fig. 1
Ao
V
Ad

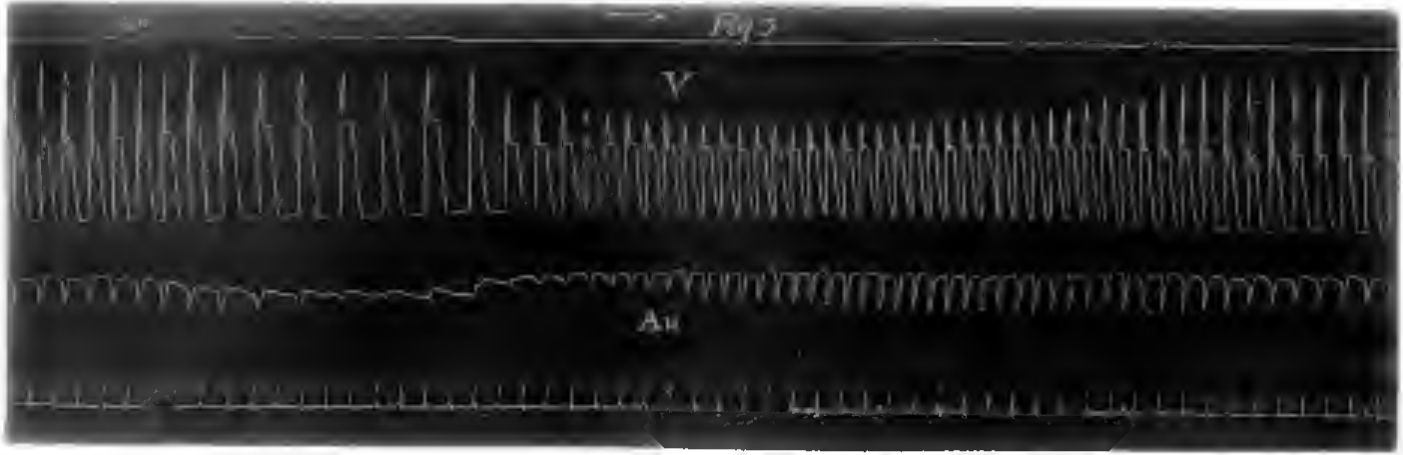
Fig 3
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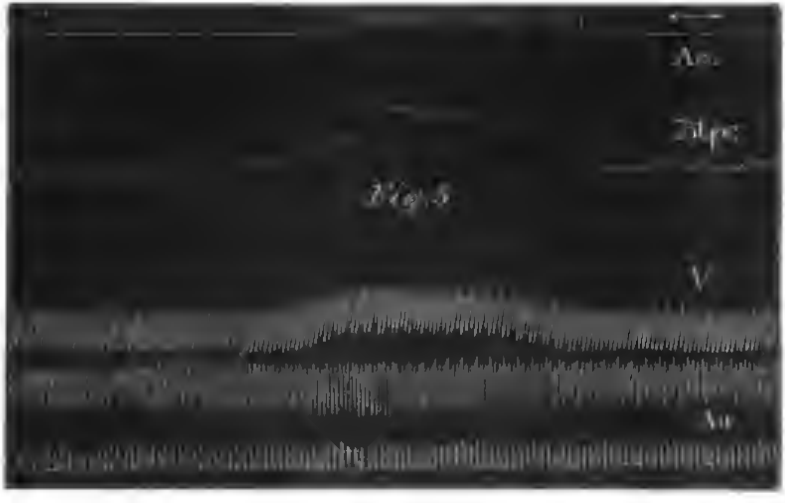
Fig. 5
V